بسمـــ الله الرحمن الرحيمـــ

فريق عمل كل الطب

يُقدمـــ

سلسلة كتب د/أحمد موافي

In Capsule Series

تم الرفع بواسطة فريق عمل كل الطب

ALLTEB MEDICAL TEAM

مجمع ومنقول من أكثر من مصدر

جزى الله خيرا كل من ساهم في هذا العمل

لا تنسونا من صالح دعائكمـــ،،

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Malabsorption Syndrome

Definition:

- Failure of absorption of one or more of the nutrient (fat, proteins, CHO, minerals..)
- **Steatorrhea** (fatty stool) remains the main mark of malabsorption.

Causes: *4*

I- Gastric Causes: 4

1-Gastrectomy. 2-Atrophic gastritis.

3-Cancer stomach → achlorhydria (↓HCl)→Bacterial Contamination of intestine.

4-Zollinger-Ellison's syndrome → hyperchlorhydria (↑HCl) → inhibits pancreatic lipase.

II- Hepato-biliary Causes : 4

1- Liver **C**irrhosis 2- **C**hronic hepatitis.

3- Biliary obstruction. 4- **B**iliary fistula.

III-Pancreatic Causes : 4

1- Chronic pancreatitis 2- **C**ystic fibrosis

3- Cancer head of pancreas. 4- **P**ancreatectomy.

IV- Intestinal Causes : (the most common)

A) Primary causes:

1- **Tropical sprue**: unknown etiology but may be:

- Bacterial, viral , parasitic infection. - Folic acid deficiency.

2- **Coeliac disease: (** *Non-tropical sprue* , *Gluten sensitive enteropathy*)

- autoimmune disorder in which there is an abnormal reaction to gluten (protein found in wheat) → Ag. Ab reaction → damage of intestinal mucosa.
- C/P : severe malabsorption syndrome , associated autoimmune diseases.
- Complications : small intestine carcinoma , ↑incidence of lymphoma.
- Investigations : Jejunal biopsy → villous atrophy , IgA antigliadin antibodies
- Treatment : The only effective treatment is lifelong gluten free diet.

B) Secodary to intestinal diseases:

1- **Short gut syndrome**: extensive intestinal resection → ↓ absorptive surface.

2- **Stagnant (*blind*) loop syndrome**: Stagnation of intestinal content e.g. strictures of small intestine , Diverticulosis → **bacterial overgrowth** → mucosal injury & nutrients utilization .

3- **Systemic diseases:** DM , amyloidosis, hypothyroidism , CHF.

4- **Infection**: -Bacterial over growth.
-TB enteritis, Giardia, Strongeloides.

5- **Inflammation**: Crohn's disease , Irradiation.

6- **Iatrogenic**: **A**ntacids , **B**iguanides , **C**holestyramine , **D**endivan .

7- **Lymphatic obstruction:**

- Lymphoma.
- Whipple's disease : (*mainly affects **MAN***)
  Malabsorption Syndrome, **A**rthritis , **N**europathy & lymphadenopathy.

Clinical picture: 4

I. General manifestations:

1- loss of weight. 2- **f**atigue.
3- **f**ever 4- clubbing of **f**ingers.

II. Intestinal manifestations:

1- **Steatorrhea**: Stool is **P**ale, **b**ulky, offensive, floats on water, **g**reasy, **g**listening.
2- **Diarrhea**: malabsorption usually results in diarrhea.
3- Audible intestinal sounds (*borborygmi*).
4- Distension, colic.

III. Nutritional deficiency:

- protein → muscle wasting, edema, recurrent infections
- Fat → loss of weight.
- CHO → hypoglycemia.

- Minerals: Iron → anemia.
  Na → Muscle cramps, hypotension.
  K → arrhythmia.
  Ca, Mg → tetany.
  Iodine → Goitre.
- Vitamins: A → night blindness, follicular hyperkeratosis.
  D → Rickets, osteomalacia.
  E → infertility.
  K → Bleeding tendency.
  C → Scurvy
  B1 → Beri – Beri.
  B2 → **g**lossitis – **g**astritis.
  B3 → **D**iarrhea, **D**ermatitis, **D**ementia.
  B6 → peripheral neuritis.
  B12 → Megaloblastic anemia.

IV. **C/P of the cause:** Coeliac disease , history of surgical operation …….

Investigation: 4

I- Biochemical investigation: 4

1- Estimation of fecal fat: (n : 6 gm/d)
- in steatorrhea → total fat is increased (> 6 gm/d)
  - split fat → intestinal disease
  - Non split fat → pancreatic disease

2- Pancreatic function tests.

3- Sugar curve : flat curve.

4- **D-xylose test**:

25 gm D xylose (*poorly metabolized sugar*) is given orally then collect urine over the next 5 hours.

- Normally : 5 hours urine should contain at least 5 gm *provided that normal renal functions.*
- In malabsorption syndrome : 5 hours will contain less than 5 gm

II- Hematological Investigations : 4

1- **Blood picture**: Anemia. (Microcytic anemia due to iron deficiency or macrocytic anemia due to vitamin B_{12} or Folate malabsorption.

2- **Plasma proteins** : hypoproteinemia.

3- **Serum electrolytes**.

4- **Prothrombin time**: may prolonged because of malabsorption of vitamin K.

III- Radiological investigations: 4

1- CT 2- MRI 3- U.S.

4- Barium meals:

◊ Dilatation of intestinal lumen. ◊ Loss of normal feathery appearance.

IV- Investigations for bacterial overgrowth :

1) 14 C- Xylose breath test :

- *14 C-Xylose is given orally then measure the 14 CO_2 in the breath.*
- In bacterial overgrowth : ↑ 14 CO2 in the breath because more bacteria will act on 14 C- Xylose.

2) Aspiration of jejunal content then culture.

Treatment:

1- Treatment of the cause:

- T.B enteritis : anti TB drugs.
- Tropical sprue : antibiotic (tetracycline) & folic acid.
- Coeliac disease :
  - The only effective treatment is lifelong gluten free diet.
  - cortisone.

2- Diet: Low fat, low fibers, non irritant diet.

3- Parentral vitamins, minerals, fluid.

4- Symptomatic treatment :

e.g. Anti diarrheal drugs : Difenoxylate *(Lomotial)* , Loperamide (*Loperazin*).

Diarrhea

Definition:

It means an increase of stool liquidity , quantity (N: 200g/d) or frequency (N: 4 motions/d).

Etiology:

I- **Etiology of acute diarrhea :**

1- **Infection**:

- **Bacterial** : **S**almonella , **S**higella , E.**c**oli , **C**holera
- **Viral** : Rota virus , Norwalk virus
- **Protozoa**: E.histolytica , Malaria , Giaradia
- **Helminthes**: Ascaris , strongyloids stercoralis.

2- **Iatrogenic**:

* Laxatives * Antibiotic * Chemotherapy
* Parasympathetic * Mg containing antacid. * allopurinol

3- **Toxins**:

* Bacterial: staph, E.coli * Lead * arscenic

4- **Diet**:

* Unripe fruit * Alcohol * Mushroom. * food allergy

5- **Nervous**: psychological stress e.g. before *internal medicine* exam. ☠

II. **Etiology of chronic diarrhea:**

1- **malabsorption syndrome**. (*enumerate its causes*)

2- Diseases of the colon :

- Irritable bowel syndrome.
- Inflammatory bowel diseases e.g. ulcerative colitis.
- Cancer colon.

3- Endocrinal causes :

- Thyrotoxicosis.
- Diabetic neuropathy.
- Addison's disease.
- Zollinger Ellison syndrome.

Mechanisms of Diarrhea:

i. **Osmotic diarrhea**: Presence of non absorbed , hypertonic substances in intestinal lumen maintain fluid & prevent absorption (e.g. lactulose, sorbitol, magnesium).

ii- **Secretory diarrhea** (*watery diarrhea*) : ↑↑ secretion of water & electrolytes into the lumen.
e.g. cholera , E.coli , ↑ VIPs (*vaso - active intestinal peptides*)

iii- **Abnormality of intestinal motility** : e.g. Thyrotoxicosis, IBS, D neuropathy.

iv- **Abnormality of intestinal mucosa** : e.g. inflammation.

Complications :

- ↓ H_2O → dehydration.
- ↓ K → hypokalemia.
- ↓ HCO_3 → Acidosis.

Diagnosis: *how to reach diagnosis of a case of diarrhea?*

1- **History analysis:**

- **Frequency**: >4/d → diarrhea
  - If the stool is of large volume & not excessive frequent → small bowel disease.
  - If the stool is of small volume & excessive frequent → large bowel disease.
- **Consistency**: - watery: inflammation
  - greasy: malabsorption.
- **Color of stool: -** bloody as in ulcerative colitis.
  - pale as in steatorrhea. – tarry as in melena.
- **Relation to meal:**
  - Osmotic diarrhea : Stops with fasting.
  - Secretory diarrhea : Continues with fasting.
- **Associated symptoms:**
  - **Abdominal pain**: all causes of diarrhea except drug induced & Thyrotoxicosis.
  - **Nausea & vomiting**: Acute infections.
  - **Fever**: infection, inflammation. - **Constipation**: IBS.

2- **Examination:**

- abdominal tenderness
- bowel sounds
- degree of general hydration
- examination per rectum: to exclude rectal mass or blood.

3- **Investigations:** **4 S**

Not every patient who presents with diarrhea needs to be evaluated with these expensive tests, watchful waiting & symptomatic therapy with oral fluid are very enough.

- **Stool examination**: - for ova, cysts & parasites.
  - stool osmolarity - fat assay.
- **Sigmoidoscopy**: if a large bowel cause is suspected.
- **Small bowel radiology**: if a small bowel cause is suspected.
- **Serological tests.**

Plus

- **Investigations Of malabsorption syndrome : (see before).**

Treatment : 3 S

- **Specific** : treatment of the cause.
- **Symptomatic**:
  - Anti diarrhea : Diphenoxylate (*lomotil*) , Loperamide (*loperazine*)
  - Anti emetic: motilium
- **Supportive**:
  - Diet: ↓ fat, ↓ irritants , light diet.
  - Treatment of complications: Fluid – K – HCO_3 .

DYSENTERY

Definition :

Diarrhea + Tenesmus + Blood + mucous in stool.

Tenesmus : *painful defecation with sense of incomplete bowel evacuation.*

Etiology :

- **A**moebic dysentery
- **B**acillary dysentery, Bilharzial dysentery.
- **C**ancer colon, rectum.
- **D**iverticulosis.
- **M**alaria
- **R**enal failure
- **G**iardia (v. rare).

Dysentery Scheme :

- Causative organism : .....
- Mode of infection : .....
- C/P : * Asymptomatic * Symptomatic * Complications
- Investigations : 4 S
- Treatment : 3 S

Amoebic dysentery

- **Causative organism** : E. histolytica (cyst form)
- **Mode of infection** : feco-oral

  (*ingestion of cysts which resist gastric acidity → transformed into vegetative form in the intestine & then passes to the colon*)

- **Clinical picture :**

  1- **Asymptomatic** : just cysts in the stool.

  2- **Symptomatic** :

  a. Acute. b. chronic

a. **Acute Amoebic dysentery :**

- **Symptom**: *One word*

dysentery : (mild diarrhea (8/d) , Tenesmus , blood & mucous in stool)

- **Signs**:
  - **Local** : Tenderness in colon especially ceacum & sigmoid.
  - **General** : - **No** fever (*superficial lesion*)
    - **No** dehydration (*mild diarrhea*)

b. **Chronic Amoebic dysentery :**

- Recurrent attacks of acute dysentery alternating with normal bowel habits.
- Abdominal pain may occur (over the caecum , appendix , transverse colon).

3- Complications:

- Local: Amoeboma (*mass in the right iliac fossa*)
- Systemic: Amoebic hepatitis (*see hepatology*).

Investigations: 4S

- **Stool examination**: cysts , mucous , blood.
- **Sigmoidoscopy**: flask shaped ulcer with intervening healthy mucosa.
- **Small bowel radiology**
- **Serological tests :** antibodies may be detected in the serum of the patient

Treatment : 3 S

- **S**pecific : ***anti amoebic***
- **S**ymptomatic
- **S**upportive

} **Like diarrhea**

Antiamoebic drugs: 4 , 3 , 2

I- **Luminal amoebicidal** : used for treatment of cysts (*chronic amoeba*)

1- Halogenated quinoline e.g. *di-Iodo-hydroxy quinoline*

- SE: Neuropathy, goiter - Dose: 500 mg t.d.s for 2 weeks.

2- Phenanthroline quinines (*Entobex*) : 500 mg t.d.s for 2 weeks.

3- Diloxanide (***Furamide***) : 500 mg t.d.s for 1 week

4- Antibiotic: paromomycin & erythromycin are directly amoebicidal.

II- Systemic drugs: (*Acute & extra intestinal Manifestation*)

Have no effect on cyst in intestinal lumen

1- Emetine: SE: Cardiotoxicity

2- Dehydro-emetine: less toxic

3- Chloroquine: used in hepatic amoebiasis.

III- Luminal & systemic (Mixed):

1- **Metronidazole** (*Flagyl*)

- Dose: 750 mg t.d.s for 10 days - SE: nausea, metallic taste.

2-**Tinidazole**: (*fasigyn*) : 2 gm daily for 5 days

Bacillary dysentery

Causative organism: G-ve bacilli (shigella)

Mode of infection: feaco-oral.

Clinical picture :

Asymptomatic

Symptomatic:

- 1st phase (1-2 days) : fever – colic – diarrhea.
- 2nd phase (1-2 weeks) :
  - **Dysentery** :
    (severe watery diarrhea (15 /d), tenesmus , mucous & blood in stool)
  - **Abdominal pain** & tenderness.
  - **General signs**: fever, malaise , dehydration.

Complication:

urethritis – arthritis – iridocyclitis – UTI (**Reiter's syndrome**)

Investigation: **4S**

- **Stool examination**: Excess WBCs & RBCs , Culture: shigella
- **Sigmoidoscopy**: diffuse inflammation with dirty yellowish pseudomembrane.
- **Small bowel radiology.**
- **Serological test.**

Treatment : 3 S

- **S**pecific :
  - **Ciprofloxacin** : 500 mg twice daily for 5 days.
  - Tetracycline: 2.5 gm in single oral dose.
  - Septrin (*Co. Trimexazole*) : 2 tab twice daily for 5 days.
- **S**ymptomatic :
  - Anti spasmodic : for severe abdominal pain.
  - Anti diarrheal drugs : are **better to be avoided** as they decrease the intestinal motility and hence decrease the wash of the organisms.
- **S**upportive : ↓ irritants , Excess fluids, vitamins.

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Bilharzial dysentery

Causative organisms:

Mainly due to ***S. mansoni*** & very rare due to *S. hematoobium*.

Mode of infection:

The parasite penetrates the skin during swimming in infected water.

Clinical picture: Incubation period : 2 months

Asymptomatic

Symptomatic:

(1) Dysentery : → Severe diarrhea alternating with constipation.
→ Tenesmus
→ Mucous & blood in stool

(2) Bleeding per rectum

(3) Pericolic tender mobile mass in left iliac fossa (*bilharzioma*)

(4) Hepato-spelenomegaly (*Egyptian splenomegaly*) **.**

Complications:

- **Hepatic bilharziasis. (……)** *see Hepatology*
- Bilharzial cor-pulmonale (……)

Investigations: 3S + 3 B

- **S**tool analysis: Bilharzial ova
- **S**igmoidoscopy: polyps or ulcers mainly in recto-sigmoid area.
- **S**erological test (by ELISA) : Schistosoma antibodies.
- **B**arium enema: polyps.
- **B**lood picture: Anemia – esinophilia.
- Investigations for **p**ortal hypertension.

Treatment:

i. **Anti bilharzial drugs:**

- **Praziquantel** (*Biltricid*)
- **Oxamniquine** (*vansil*) :20 mg/kg orally for 3 doses (against S. mansoni only)
- Niridazole (*ambilhar*) : 25 mg/kg/d orally for 7 days.
- Metrifonate : 10 mg/kg orally.(S. hematobium)
- Mirazid (*Commiphora ext.*): Herbal drug , dose : 12 capsules over 6 days.

Praziquantel :

- Mechanism of action : Ca influx into the worm → marked contraction & spastic paralysis of the worm.
- Dose: 40mg / kg in two divided dose.
- S/E: headache, abdominal pain , nausea, vomiting, pruritis, fever, elevation of liver enzymes.

NB :

- Antibilharzial drugs must be given early before tissue fibrosis.
- Repeated courses may be needed as the worms migrate into the liver & return back to the periphery
- Fever & vitamins deficiencies must be treated before starting therapy.

ii. **Endoscopic polypectomy.**

iii. **Treatment of the complications** e.g. Portal hypertension.

iv. **Symptomatic & supportive treatment.**

VOMITING

Definition:

forceful expulsion of gastric content into the mouth.

Mechanism of vomiting reflex:

→ stimulus → centre → response (vomiting)

- **stimulus**: - peripheral (*GIT*).
  - Central e.g.: ↑ ICT.
  - Metabolic e.g.: uremia.
- **centre** : vomiting centre in the medulla.
- **Response**: vomiting.
  - ◊ Pylorus is closed. ◊ cardia is opened.
  - ◊ contraction of the diaphragm& abdominal wall muscles.

Etiology:

I- Peripheral causes: (GIT causes)

- Gastritis - peptic ulcer – cancer stomach
- Hepatitis - pancreatitis - Appendicitis
- Cholecystitis - peritonitis
- pyloric obstruction – intestinal obstruction.

II- Central causes:

- Psychic : bad smell, sight.
- Anorexia nervosa.
- ↑ ICT: Tumor , Hemorrhage.
- Pain: migraine, MI, renal colic.

III- Metabolic causes:

- Renal failure. - Liver cell failure.
- Acidosis (DKA).
- Electrolyte: ↑ Ca, ↓ Na, ↓ or ↑K

IV- Iatrogenic: **MAD**

- **m**orphine. - **A**lcohol. - **D**igitalis.

V- Pregnancy.

Complications of severe vomiting :

- dehydration.
- alkalosis (tetany).
- cerebral hemorrhage .
- pulmonary aspiration.

Diagnosis of a case of vomiting:

I-history analysis II-physical examination III- Investigation

I- History analysis :

(1) Time of vomiting:

* Early morning :
  - □ Pregnancy □ ↑ ICT □ Alcohol
* During meal : Esophageal cause.
* After meal by:
  - □ ½ h : Gastric ulcer.
  - □ 2-4 h : Duodenal ulcer.
  - □ > 8h : Intestinal obstruction.

(2) Associated nausea: upper GIT causes.

- No nausea: ↑ ICT

(3) Associated Abdominal Pain : (GIT causes)

- e.g. peritonitis , intestinal obstruction , pancreatitis , cholecystitis
- painless vomiting : Neurological causes.
- pain relieved by vomiting : Gastric ulcer.

(4) Associated headache: ↑ ICT.

(5) History of drug intake : MAD

(6) Biliary symptoms:

Jaundice , Pruritise , Abdominal Pain , change of color of urine & stool.

(7) Frequency: Persistent in **p**eritonitis.

II- Examination of vomiting:

- **Amount** :huge in pyloric obstruction .
- **Color :** coffee ground as in hematemesis.
- **Content** :
  - undigested food: pyloric obstruction.
  - bile: below ampulla of vater
  - blood: ulcer, cancer, Mallory weiss $
  - F.B. : gall stone.
- **Odour**: offensive as in intestinal obstruction , malignancy.

III- Investigations: investigations for the causes.

Treatment :

- **S**pecific: for the cause.
- **S**ymptomatic:
  - Antiemetic : motilum , primpran.
  - Antidiarrheal drugs.
- **S**upportive : correct dehydration & electrolyte imbalance .

CONSTIPATION

Definition: Infrequent passage of hard stool .

Causes: 7 **I**

i. **I**mmobility (prolonged rest in bed).
ii. ↓ **I**ntake of fluid & fibers .
iii. **I**rritable bowel syndrome .
iv. ↓ **I**ntestinal motility : hypothyroidism , DM , ↑ Ca , ↓ K .
v. **I**atrogenic: codeine , morphine , anticholinergic, aluminum hydroxide.
vi. **I**nflammation : ulcerative colitis , diverticulitis .
vii. **I**ntestinal obstruction : cancer colon , stricture .

INFLAMMATORY BOWEL DISEASE

(Crohn's disease & ulcerative colitis)

Etiology:

- Is still questionable.
- Most probably autoimmune. (The local T cells are angry with something in the food)
- Genetic factor: play a great role.
- Infections (T.B, measles): not confirmed.
- Psychological factor may play a role.

	Crohn's disease	Ulcerative colitis
Pathology:		
- Site:	Affects any part of GIT from mouth to anus, esp. terminal ileum (70%).	- Limited to colon & rectum. - Terminal ileum can be affected in 5% only
- Layers	All three (*transmural*)	- Mucosa only
- Granuloma	Yes in (30%)	No
- Crypt abscesses	No **N.B: finding of granuloma = crohn's disease**	Yes (30%) **N.B: finding of crypt abscesses = ulcerative colitis**
- Fibrosis	Server	No
- Pattern of colonic involvement	Skip lesions	Continuous involvement
C/P:		
- Sex	Equal	Slightly more in ♀ ☺
- Age	No specific age	30-40 Y
- Smoking	↑↑ incidence in smokers	↓↓ incidence in smokers (*80% non smokers*)
- Abdominal Pain:	Prominent	Less prominent

	Crohn's disease	Ulcerative colitis
- Diarrhea	Yes	Yes (*severe & sometimes dysentery*)
Bleeding per rectum	Uncommon	Common
Anal & oral lesions	Yes	No
Bowel obstruction	May be	No
Malabsorption syndrome	May be	No
Intestinal fistula	May be	No
B_{12} deficiency	May be	No
Carcinoma risk	Minor	High
Systemic (extra intestinal manifestations)	• Skeletal : Arthritis, • Skin:erythema nodosa, pyoderma gangrenosa ♫ • Liver: fatty liver – sclerosing cholangitis • Lung : interstitial pulmonary fibrosis. • Eye: uveitis, iritis	
Investigation		
- Radiological :	- deep ulcers (*cobble stone*) - areas of narrowing in the ileum. - Skip lesion - Fistula & strictures.	- shortened colon , absence of hustra (*lead pipe sign*) - pseudo polyposis 15%.
- Colonoscopy & biopsy	- Show involvement of the entire bowel wall with granuloma formation.	- Limited to the mucosa & may have crypt abscesses.

Treatment:

Medical treatment :

1- Sulfasalazine: reduce inflammation especially in ulcerative colitis.

1 gm /6 h reduced to a maintenance dose of 2 gm/ day for 2 years after remission .

2- Steroid (prednisone) : 60mg/d. This is the main treatment for active disease

3- Immuno Suppressants: Azathioprine (*Imuran*).

4- Symptomatic :

- Antibiotics for 2ry infection : Metronidazole & ciprofloxacin.

- Anti diarrheal drugs.

Surgical treatment : (Indications)

- Failure of medical treatment. - Severe complications

IRRITABLE BOWEL SYNDROME (IBS)

Definition:

It's a functional disturbance of colonic motility with no organic cause .

Etiology : unknown , psychological disturbance play a role.

Clinical picture: long history with long free interval

- Recurrent pain :over any part of the colon (especially in left iliac fossa).
- The pain is ↑↑ by food & ↓↓ by defecation .
- There is constipation or diarrhea .
- Abdominal distension is common .
- Patient with IBS often complain of anxiety , depression & tension headache.

Investigations : (to exclude common diagnosis)

- no +ve finding .

Treatment :

- ☺ Reassurance .
- ☺ Diet : high fiber diet & bran , Avoid coffee , tea , smoking .
- ☺ Mild sedative , antispasmodics .
- ☺ Tegaserod (***Zelmac***) *one tablet / 12 h : activate serotonin type 4 receptors in GIT* .

DYSPEPSIA

Definition:

Any abdominal discomfort related to meal e.g. heartburn , epigastric pain , distension , nausea,…….

Causes of dyspepsia :

- **GIT causes :** - reflux esophagitis - peptic ulcer - cholecystitis
  - pancreatic disorders - hepatic diseases.
- **Systemic diseases :** Renal failure , hypercalcemia.
- **Drugs :** NSAID , cortisone .
- **Functional dyspepsia :** it's a persistent dyspepsia with no organic cause (–ve endoscopy & the patient is Helicobacter pylori – negative).

Diagnosis :

History analysis:

- What type of discomfort does the patient feel ? e.g. heartburn , distension…

- Is this discomfort related to meal ?

- Time to meal :

- **During meal :** Esophageal cause : Reflux esophagitis**.**
- **½ H after meal :** Gastric cause e.g. Peptic ulcer .
- **2-3 H after meal :** Duodenal cause e.g. Duodenal ulcer.
- **8 H after meal :** Intestinal cause : obstruction .

- Type of meal :

☺ Fat : Cholecystitis ☺ Meat : Cancer stomach . ☺ Starch : Gastric ulcer .

-Associated symptoms .

Investigations : for the cause

Patients with persistent dyspepsia need to be referred for **endoscopy** if they are **over 50 years old** , or they have associated weight loss , dysphagia , GIT bleeding or previous gastric surgery .

Treatment :

☺ **Diet :** eat little & often , Avoid fatty food, tea, coffee , smoking, alcohol.

☺ **Symptomatic treatment .**

☺ **Treatment of the cause .**

Diseases of Esophagus

- The esophagus is 25 cm long connecting the pharynx to the stomach .
- The mucosa is lined with squamous epithelium .
- The esophagus is protected from acid damage by a number of defenses including the lower esophageal sphincter pressure , gravity ,salivary bicarbonate & esophageal bicarbonate secretion .

Hiatus Hernia

- Occurs when part of the upper stomach herniates through the diaphragm into the chest .

– It's extremely common , especially with increasing age & obesity .

– **There are 2 types :**

1- Sliding 80 % :

- May be asymptomatic .
- May cause acid reflux , aspiration. - Bleeding may occur.
- **Investigations** : Barium swallow . upper endoscopy .
- **Treatment** :□ Weight reduction (Meals should be small)
  - □ Sleeping in semi-sitting position .
  - □ H_2 blockers & proton pump inhibitors (*omeprazole*)
  - □ Prokinetic drug : dompridone (motilium).
  - □ **Surgery** : indicated in resistant cases (*fundoplication*)

2- Rolling 20 % : (para-esophageal hernia)

- Here , a small part of the fundus of the stomach rolls up alongside the esophagus through the hiatus .
- The gastro-esophageal junction is not affected so , there is no reflux
- may obstruct or strangulate .
- A big hernia may lead to mediastinal syndrome .
- Surgery is indicated in severe cases .

Esophageal Achalasia

Definition : It's a condition of unknown etiology resulting in abnormal peristalsis and lack of relaxation of the lower esophageal sphincter (LES) .

Clinical picture :

- Intermittent dysphagia : to fluid & to solid food .
- Regurgitation , chest pain , infection ,mild weight loss may occur.

Investigation :

Barium swallow:-Esophageal dilatation with a smooth distal (bird's beak).
-Absence of gases in the fundus of the stomach.

Upper endoscopy .

Manometry : - high pressure in lower segment .
- Loss of peristalsis .

Treatment : Endoscopic dilatation or surgical myotomy .

Gastro-Esophageal Reflux Disease

(GERD)

Definition : Reflux of gastric contents into the esophagus.

Etiology & pathogenesis : (Failure of anti reflux mechanisms)

1- Decrease tone of lower esophageal sphincter (LES) .
2- Decrease esophageal clearance of acid due to poor esophageal peristalsis.
3- Delayed gastric emptying .
4- Hiatus hernia may aggravate the condition .

Predisposing factors :

♦ obesity . ♦ smoking . ♦ alcohol . ♦ coffee .
♦ large meals (especially late at night) .
♦ drugs : Ca antagonist , theophylline , anticholinergic & nitrates .

Clinical picture :

1- Heartburn : the main symptom .

2- Chest pain : - Epigastric & retrosternal (simulating angina) ☹

- Increased on lying down & relieved by antacid .

3- Odynophagia : painful swallowing .

4- Dysphagia : due to disturbed motility .

5- Pulmonary : cough , asthma , aspiration pneumonia may occur .

6- Hematemsis & melena : due to mucosal inflammation or ulceration.

7- Laryngeal irritation .

8- **Barrett's esophagus** : (May occur in long standing acid reflux) .

- The normal squamous epithelium is replaced by the columnar gastric epithelium
- It's a premalignant leading to adenocarcinoma .

Investigations: (GERD is a clinical diagnosis)

- **Esophageal PH monitoring** (gold standard for diagnosis) . MCQ

- Endoscopy. - Manometry. - Barium swallow : it may show hiatus hernia .

Treatment :

1- Diet :

☻ Limit intake of food & drink that reduce lower esophageal sphincter pressure : fatty food , acidic foods , onions ,chocolate, coffee , alcohol .

☻ Avoid heavy meals especially before sleep . ☻ Weight reduction . ☻ Stop smoking .

2- Postural therapy :

☻ Raising the head of the bed at night .

☻ Avoid lying down after eating .

☻ Remain upright at least 2h after eating .

3- Drug therapy :

☻ *↓ gastric acidity :*

- Proton pump inhibitors : Omeprazole , *most potent single agent*.
- Antacid . - H_2 blockers : Ranitidine .

☻ *↑ esophageal peristalsis :* Domperidone (motilium)

4- Surgical & Endoscopic therapy : In resistant cases .

DYSPHAGIA

Definition: **Difficulty in swallowing ,**with a sensation of sticking or obstruction of the passage of food through the mouth , pharynx or the esophagus .

Causes :

1- Transfer dysphagia :

(alteration of neuromotor mechanism of the oropharyngeal phase, neurological causes)

- □ Bulbar palsy .
- □ Myasthenia gravis .
- □ Stroke .

2- Transit dysphagia : (motor disorders , abnormal peristalsis)

- □ Achalasia .
- □ Scleroderma .
- □ Esophageal spasm .

3-Obstructive dysphagia: (mechanical narrowing of the esophagus)

- □ Esophageal stricture .
- □ Cancer esophagus .
- □ All causes of mediastinal syndrome : Goitre , bronchogenic carcinoma , LN

4- Odynophagia : (painful swallowing)

- □ Inflammation of (mouth, tongue, pharynx, tonsils or esophagus)

5- Hysterical : **(Globus hystericus)**

Diagnosis of a case of dysphagia :

History analysis : **Ask about :**

- Onset , Course , Duration.
- Type of food (solid or liquid)
- Painless or painful .
- Associated symptoms .
- Site of obstruction .

Examples :

Transfer dysphagia :

♥*Onset* : Acute ♥ *Course* : variable .

♥*Type of food* : more to **liquid** .

♥*Associated symptoms*: Nasal regurgitation , Dysphonia, neurological manifestations.

Transit dysphagia :

e.g. achalasia

♥ Intermittent & may progress –long duration.

♥ **Liquid** > solid.

♥ Relieved by repeated swallowing.

♥ **Associated symptoms** : Heartburn precipitated by eating , no marked loss of weight .

e.g. scleroderma

♥ Intermittent course .

♥ Both solids & liquids .

♥ Associated symptoms : Nocturnal cough , regurgitation .

NB : Dysphagia that worsens on ingestion cold liquids & improves with warm liquids suggests a motor disorder(transit dysphagia).

Obstructive dysphagia :

e.g. cancer esophagus

♥ Progressive – short duration .

♥ Dysphagia to **solids** that may progress to include liquids.

♥ Associated symptoms : weight loss , chest & back pain .

Investigations:

♥ **Barium swallow**: (***initial diagnostic step after history & examination***)

♥ **Endoscopy .**

Stomach & duodenum

Physiological aspect :

- HCL is secreted by the parital cells of the stomach through the action of hydrogen-Potassium ATPase (proton pump)
- **Function of HCL :**
  - Converting pepsinogen to pepsin , initiating the first stages of protein digestion.
  - Antibacterial barrier.
- **HCL secretion is stimulated by :**
  - Vagus (directly & via increasing gastrin)
  - Gastrin.
  - Histamine.
- **HCL secretion is inhibited by :** MCQ
  - Somatostatin
  - Secretin
  - Cholecystokinin

N.B.

- Parital cells → secret HCL & intrinsic factor.
- Peptic cells (chief) → secret Pepsin.
- G cells → secret Gastrin.

Peptic ulcer

Definition :

- Ulceration of mucosa which is exposed to acid peptic juice.
- The following sites are affected :
  - Duodenum.
  - Stomach.
  - Jejunum (after gastrojejunostomy)
  - Esophagus.
  - Meckel's diverticulum (contains ectopic gastric tissue)

Etiology :

It occurs when stomach acid penetrates the stomach and/or duodenal lining.

- **It's a mix of :**
  - Hyperacidity : duodenal ulcer.
  - Decreased mucosal resistance : gastric ulcer.

1- **Helicobacter pylori infection** (duodenal > gastric)

2- **Traditional NSIDs** : (gastric > duodenal).

3- Rare causes : Zollinger Ellison syndrome (↑ acid production)

~ **S**moking , **S**picy , **S**tress are no longer thought to be a cause of ulcers , but they can make ulcers worse (*aggravating factors*)

~ Smoking → ↑ acid & ↓ protective prostaglandin.

1- Helicobacter pylori :

- Responsible for the majority of peptic ulcer (90% of DU , 70% of GU).
- H.pylori is a gram -ve spiral bacillus transmitted by feco-oral or through mouth to mouth such as kissing.
- Many people have H.pylori infection, but not everyone who has an infection will develop a peptic ulcer.
- Pathogenesis of H.pylori :
  - ↓ somatostatin.
  - ↑ gastrin release.
  - Produce cytotoxins causing mucosal inflammation.

2- Non steroidal anti-inflammatory drugs (NSAIDs) :

- NSAIDs act by inhibiting cyclo-oxygenase enzyme (COX) leading to decrease prostaglandin → mucosal erosions & ulceration.
- Notice that prostaglandin play a big role in gastric protection.

NB : All people at high risk of developing peptic ulcer should use COX-2 inhibitor rather than one of the traditional NSAIDs (*COX-1 inhibitor*) , however studies have shown that COX-2 inhibitors appear to increase the risk of heart attacks & stroke with long term use , so most doctors now use a traditional NSAIDs plus a strong acid inhibitor.

Disorder associated with peptic ulcer :

- Cirrhosis.
- CRF.
- COPD.
- Coronary diseases.

NB : *Peptic ulcer is more common in cirrhotics due to ↓ destruction of gastrin & histamine by the liver & ↓ mucosal resistance due to congestive gastropathy caused by portal hypertension.*

Clinical picture :

Symptom : Epigastric pain

- Character : burning , gnawing or dull ache.

- Site :

- DU : above the umbilicus & to the right of the midline.
- GU : epigastric & in the midline.

Sudden onset of severe generalized pain may indicate perforation.

- Duration : variable from few minutes to several hours.

- Relation to meal :

- DU : 2 - 3 h after meals.
- GU : precipitated by food ½ h after meals

So, in DU : pain awakes the patient from sleep (*the most important symptom*)

- Reliving factors :

- DU : antacid or food , so appetite ↑ (patient eats frequently to relief pain)
- GU : fasting , vomiting (some patients learn to induce vomiting for pain relief)

- Associated symptoms :

anorexia , vomiting & weight loss in GU.

Signs : may be -ve

- **Localized tenderness** at the site of ulcer (severe tender suggests a perforation)
- Physical examination is critically important for discovering evidence of ulcer complications.

Complications :

- GIT **b**leeding : most common complication (15%) , **may be the first symptom.**
- **P**erforation : The second most common complication.
- **P**yloric obstruction.

Investigations :

- **Endoscopy** : The best to detect the ulcer.

> - *Endoscopic biopsy should be taken in all gastric ulcer to differentiate between benign & malignant ulcer.*
> - *Malignant gastric ulcer is mostly malignant from the start.*

- Occult blood in stool.
- Investigations for H. Pylori : antibodies , culture & sensitivity test.

Treatment :

i. Diet :

- Frequent small meals.
- Avoid spicy food , smoking , alcohol.

ii. Medical treatment :

1. Antacid : (*symptomatic relief in mild cases*)

   e.g. mixture of Aluminum & Mg hydroxide (*Maalox*)

2. H_2 blockers : (for 4-8 weeks)

   They block H_2 receptors → reduction of acid secretion.

   - Cimetidine : 200 mg four times/d . SE : reversible gynecomastia & impotence.
   - **Ranitidine** (*Zantac*) : 150 mg twice /d or better 300 mg at bed time.
   - Famotidine : 20 - 40 mg/d.

3. Proton pump inhibitors (PPI) : (**for 4 - 6 weeks**)

   - They inhibit H - K ATPase enzyme (proton pump) → ↓ H^+ secretion → ↓ HCL secretion.

   - Omeprazole (Losec) : 20 mg / d.
   - Lansoprazole : 30 mg / d.
   - Pantoprazole : 40 mg / d.

4. Sucralfate: (for 4 -6 weeks)
   - It's a mucosal protective agent (*coating agent*)
   - Dose : 1 gm / qid.
   - Not given with antacid.

5. Anticholinergic drugs:

   Pirenzepine (*gastrozepine*) : selective M_1 blocker.

6. Eradication of H. Pylori: (*triple therapy for 2 weeks*)
   - No single agent is effective in eradication this organism.
   - Triple therapy is used for 2 weeks **e.g.**
     - Omeprazole 20 mg twice daily.
     - Clarithromycin (*Klacid*) 500 mg twice daily.
     - Metronidazole 500 mg/d or Amoxycilline 1 gm /d twice daily.
   - Eradication of H. Pylori may lead to dramatic decrease in ulcer recurrence to less than 5%.

7. Avoid NSAIDs:

   If NSAIDs must be continued , either use selective COX-2 inhibitors as Lomoxicam (*Xefo*) or use traditional NSAIDs plus a strong acid inhibitor.

iii. Surgical treatment :

- **Indication :**

  Recurrent bleeding , Perforation , Pyloric obstruction , Malignancy.
- **Operations :** Partial gastrectomy , Vagotomy.

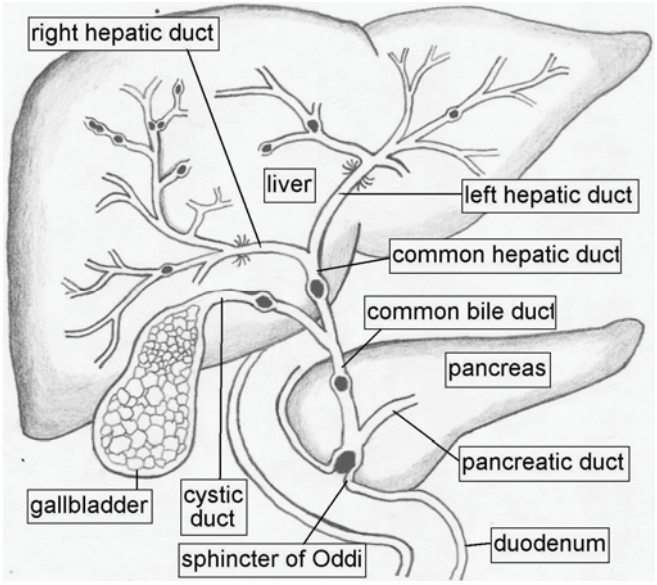
iv. Treatment of complications :

- **Bleeding :**
  - Ranitidine or Omeprazole infusion.
  - Endoscopic injection of adrenaline.
  - Blood transfusion.
- **Perforation :** IV fluid , analgesic & antibiotic then surgery.
- **Pyloric obstruction :** surgery.

Gall bladder & Biliary system

Anatomy of Biliary system :

- The right & left hepatic duct arise from the right & left lobes of the liver & unite to form the common hepatic duct which is joined by the cystic duct from the gall bladder to form the common bile duct (CBD) which enters the duodenum (*often after joining the main pancreatic duct*) through the ampulla of vater.
- The function of the gall bladder is to store bile that is produced in the liver before the bile is secreted into the intestines.
- Approximately 1 litre of bile is secreted by the hepatocytes each day .Half of this drain directly into the duodenum & the reminder is stored in the gallbladder . Cholecystokinin then stimulates its release.



Gall stones

Etiology :

1) **Cholesterol stones :**
   Cholesterol is kept soluble by the presence of bile salts so , if bile salts decrease or cholesterol increases , cholesterol stones will occur.
2) **Pigment stones :** rare
   Excessive production of bile pigments in bile e.g. chronic hemolysis.
   80% gallstones are a mixture of cholesterol & bile pigment (*mixed stones*)
3) **Stasis :**
   - Estrogen : due to GB wall relaxation.
   - Long term parenteral nutrition : lack of oral intake → ↓ cholecystokinin that stimulate the GB contraction.

Risk factors for stone formation :

- Fatty (obesity) - Forty (increasing age) - Fertile (multi parity) - Female.
- DM - Drugs : contraceptive pills , Octreotide.

NB : *Stones may form in the CBD even after cholecystectomy.*

Clinical picture : gall stones are present in 10 - 20% of population

- **A**symptomatic : 80% of cases.
- **A**scending cholangitis (*bacterial infection*)
- **P**ancreatitis.
- **B**iliary colic.
- **C**holecystitis.
- **Obstructive jaundice** if duct obstruction.

Investigations :

- **US :** should be performed routinely in patients suspected of having gallstone disease
- **ERCP :** is the best technique for diagnosing common bile duct stones, but it is far less sensitive in detecting stones in the gallbladder than US.

NB : The major component of most gallstones is cholesterol, which is not radio-opaque. This explains why many gallstones do not show up on a plain X-ray; only stones with high calcium content are radio-opaque and visible.

Treatment :

- **Cholecystectomy is a definitive treatment** *(either surgical or by laparoscope)*
- Shock wave lithotripsy.
- Medical treatment : through the oral administration of bile acids, such as *ursodeoxycholic acid* to dissolve cholesterol stones.

Acute cholecystitis

Etiology & pathophysiology :

- It's usually caused by obstruction of the cystic duct by stones.
- Begins with sterile inflammation , then becomes infected (E.coli , Strept. fecalis)
- GB may distended with mucous (*catarrhal cholecystitis*) or pus (*suppurative cholecystitis*) .
- Rarely, acute gangrenous cholecystitis may develop.

Symptoms : Severe pain

- This pain starts usually in the right upper quadrant or epigastrium.
- May radiate to the back or right shoulder.
- Biliary pain typically is gradual in onset and lasts hours.
- Nausea & vomiting may occur.

Signs :

- Fever.
- Obstructive jaundice may occur due to edema or stone in CBD.
- Murphy's sign : ask the patient to take deep inspiration while palpating the G.B. area → the patient catch his breath due to sudden pain.
- Boas's sign : there is an area of hyperesthesia between the 9^{th} and 11^{th} ribs posteriorly on the right side.

Investigations :

1- Laboratory :

- Leucocytosis.
- Serum bilirubin , AST , ALT & alkaline phosphates : may be elevated.

2- Radiological : US , Radioisotope scanning.

3- ECG : done as a routine especially in the elderly patients to exclude IHD.

Treatment :

Medical :

- Bed rest , IV fluid.
- Analgesics : pethidine (no morphine as it induces spasm of the sphincter of Oddi)
- Antibiotic : 3^{rd} generation cephalosporin.
- Antiemetic.

Surgical :

- If the attack subsides , cholesystectomy is done after 1- 3 months.
- Urgent surgery : in cases with complications.

Chronic Calculus Cholecystitis

Etiology : It's almost always associated with the presence of gall stones.

Clinical picture :

- There are recurrent attacks of right upper abdominal pain.
- Dyspepsia , local tenderness & Murphy's sign is usually present.

Investigations : US shows thick G.B. wall & gall stones.

Treatment : Cholecystectomy.

Acute Pancreatitis

Etiology :

- **gall stones → obstruction of pancreatic duct (*the most common cause*)**
- Alcohol.
- Viral infection e.g. mumps, Coxsackie B.
- Hyperlipidemia.
- Hypercalcemia.
- Idiopathic.

NB : Gall stones → back regurge of trypsin enzyme (*autodigestion*)

Symptoms :

- Abdominal Pain: epigastric pain radiating to the back.
- Nausea & vomiting.

Signs :

Unexpectedly, abdominal signs are minimal in comparison with the severity of pain.

- Tenderness & rigidity : at first in the upper abdomen, later on become generalized.
- Cullen sign: umbilical ecchymosis.
- Grey turner's sign: ecchymosis in flanks.

Complications:

- Multiple organ failure (*MOF*) : HF, Renal failure, Respiratory failure.
- **A**bscess.
- **P**ancreatic ascites.
- Hypo **C**alcemia.
- **D**IC.
- **D**iabetes mellitus.
- Obstructive jaundice : due to edema of the head of the pancreas.

Poor prognostic indicators :

mnemonic : Pancreas

- **P**aO2 < 60mmHg .
- **A**ge > 55 years.
- **N**eutrophils: WCC > 16,000 /mm^3.
- **C**alcium < 8mg %.
- **R**enal: Urea > 100mg %.
- **E**nzymes: LDH > 350 ; SGOT > 250 U.
- **A**lbumin < 3gm%
- **S**ugar: Glucose > 200 mg % .

Investigation:

1. Pancreatic enzymes :
   - Amylase (N : 75-150 somogyi unit) : ↑↑ (in blood , urine , peritoneal fluid)
   - Lipase : ↑↑ , more specific than amylase.

NB : *Serum amylase is raised in other conditions e.g. perforated peptic ulcer , ectopic pregnancy , alcoholics , salivary gland disease .....)*

2. Peritoneal aspiration : contains high amylase.
3. Blood picture : leucocytosis , anemia in hemorrhagic pancreatitis.
4. X ray, US, **CT** : gall stones , swollen pancreas , calcification.
5. ECG : to exclude myocardial infarction.

Treatment :

- Nasogastric suction.
- IV nutrition.
- Antibiotics.
- Analgesics : pethidine (no morphine as it induces spasm of the sphincter of Oddi)
- Somatostatin infusion.
- Treatment of complications e.g. Respiratory support , cortisone.
- Surgery for pancreatic abscess & ERCP may be needed.

Prognosis : death rate is about 10%.

Chronic Pancreatitis

Chronic pancreatitis is an inflammatory condition characterized by irreversible damage to the exocrine & later to the endocrine tissue of the pancreas.

Etiology : **Like Acute pancreatitis but :**

- **Chronic alcoholism** is the commonest cause.
- Gall stones are unlikely to cause chronic pancreatitis.

Clinical picture : Triad of :

i- Recurrent abdominal Pain: radiate to the back.

ii- DM

iii- Steatorrhea *(malabsorption $)*

Investigation:

- Like acute pancreatitis.
- CT is the most sensitive for detection of pancreatic calcification.
- ERCP : shows irregular dilation and structuring of the pancreatic ducts.

Treatment :

1. Stop alcohol
2. Pancreatic enzyme replacement : taken as enteric coated tablets.
3. symptomatic treatment :
    - pain : Analgesics.
    - Treatment of DM.
    - Treatment of steatorrhea.

Cancer Head of Pancreas

- painless obstructive jaundice.
- loss of weight.
- Anorexia.

Cancer Body of Pancreas

- Pain: radiate to back.
- Jaundice: rare.

Tuberculous peritonitis

Etiology :

- **Primary :** Occurs in children by ingestion of infected milk *(bovine bacilli)*
- **Secondary :** Reactivation of a Tuberculous focus in the abdomen e.g. LN , Pulmonary TB.

Pathological type :

1- **Ascitic type :** Accumulation of free fluid in the peritoneum with no adhesion .

2- **Loculated type :** The peritoneal cavity is divided by fibrous adhesions into loculi containing ascetic fluid.

3- **Adhesive type :** Marked adhesion of the intestinal loops.

Clinical picture :

usually gradual

Symptoms :

- General symptoms : night fever, night sweating, loss of appetite, loss of weight.
- Constipation or diarrhea (*malabsorption syndrome*).
- Abdominal pain & distension.

Signs :

2 T , 2 P

- **T**oxemia : toxic face , fever ,….
- **T**enderness with mild rigidity.
- **P**alpable masses (rolled omentum)
- **P**ercussion may reveal shifting dullness.

Complications :

- Intestinal obstruction.
- Intestinal fistula.
- Spread of tuberculosis.
- Amyloidosis.

Investigations :

i. Abdominal Ultrasonography : detect mild ascites , LN.

ii. X ray : calcified mesenteric LN.

iii. Aspiration of ascetic fluid :

- Straw colored. - Rich in lymphocytes & proteins. - Z.N. or PCR for the organism.

iv. Laparoscopy or Laparotomy.

Treatment :

Anti - Tuberculous drugs see chest

DD of epigastric pain :

Esophageal : - Esophageal spasm - Hiatus hernia. - Esophagitis. - Cancer.

Gastro-duodenal : - Gastritis - Peptic ulcer. - Gastric carcinoma.

Gall bladder : - cholecystitis. - Gall stones.

Pancreas : - pancreatitis. - Cancer pancreas.

Hepatic : - Hepatitis. - Congested liver. - Hepatoma.

Cardiovascular : -Angina - MI. - Pericarditis. - Aortic dissection.

Chest : -Pneumonia. -Pleurisy. - Repeated cough.

Nervous : - Herpes zoster. - Neurosis.

Medical conditions that mimic acute abdomen :

- Myocardial infarction.
- Rheumatic fever.
- Dissecting aortic aneurysm.
- Congestive heart failure : right upper abdominal pain due to congested liver.
- Diabetic ketoacidosis.
- Tetany.
- Hemolytic crisis.
- Henoch Schonlein purpura.
- Acute intermittent porphria.
- Acute pancreatitis.
- Renal failure.
- Hypoglycemia.
- Familial Mediterranean fever. (*FMF*)
- Vasculitis.
- Irritable bowel syndrome (*IBS*).

Acute pain in the upper abdomen

Oesophagitis	***Suggested by:* retrosternal pain, heartburn.**
	Confirmed by: ***oesophagogastroscopy***.
Acute coronary syndrome (unstable angina or infarction)	*Suggested by:* chest tightness or pain on exertion. *Confirmed by:* ***exercise ECG*** , cardiac enzymes ,***coronary angiography*** .
Hiatus hernia	*Suggested by:* heartburn, worsens with stooping or lying, relieved by antacids.
	Confirmed by: ***oesophagogastroscopy, barium meal***.
Gastritis	*Suggested by:* epigastric pain, dull or burning discomfort, nocturnal pain *Confirmed by:* ***oesophagogastroscopy, barium meal and pH study***.
Gallstone colic (with no acute inflammation or infection)	*Suggested by:* jaundice, biliary colic, pain in epigastrium or right upper quadrant radiating to right lower scapula. No fever.
	Confirmed by: ***ultrasound of gallbladder and biliary ducts***.
Acute cholecystitis	*Suggested by:* fever, guarding and positive Murphy's sign (abrupt stopping of inspiration when the palpating hand meets the inflamed gall bladder descending with the liver from behind the sub-costal margin on the right side,but not on the left side). ↑ WBC. *Confirmed by:* ***ultrasound gallbladder and biliary ducts***.
Duodenal ulcer	*Suggested by:* epigastric pain, dull or burning discomfort, typically relieved by food, nocturnal pain.
	Confirmed by: ***oesophagogastroscopy, barium meal and pH study:*** (*Helicobacter pylori* often present in mucosa or serology).
Gastric ulcer	*Suggested by:* epigastric pain, dull or burning discomfort, typically exacerbated by food, nocturnal pain. *Confirmed by:* ***oesophagogastroscopy, barium meal and pH study***.
Gastric carcinoma	*Suggested by:* marked anorexia, fullness, pain, large lymph node in the left supraclavicular fossa.
	Confirmed by: ***upper GI endoscopy with biopsy***.
Pancreatitis	*Suggested by:* pain radiating straight through to the back, better on sitting up or leaning forward. *Confirmed by:* ***↑ serum amylase, CT pancreas***.

Acute central abdominal pain

Small bowel obstruction	*Suggested by:* vomiting, constipation with complete obstruction.
	Confirmed by: ***X*** ray shows small bowel loops and fluid levels.
Crohn's disease	*Suggested by:* chronic diarrhoea with abdominal pain, weight loss, palpable RLQ mass or fullness, mouth ulcers.
	Confirmed by: ***colonoscopy with biopsy, barium studies*** showing skip lesions.
Mesenteric artery occlusion	*Suggested by:* vomiting, bowel urgency, melaena, diarrhoea.
	Confirmed by: ***mesenteric angiography, exploratory laparotomy***.
Abdominal aortic dissection	*Suggested by:* tearing pain , shock , hypotension and peripheral cyanosis.
	Confirmed by: ***ultrasound or CT abdomen***.

Acute lateral abdominal pain

Pyelonephritis	*Suggested by:* pain in loin (upper lateral), rigors, fever, vomiting, frequency of micturition, renal angle tenderness.
	Confirmed by: ***CBC:*** leucocytosis.***urinalysis :*** pyuria, urine culture and sensitivity
Renal calculus	*Suggested by:* renal colic mainly in loin (upper lateral), hematuria.
	Confirmed by: ***urinalysis, renal ultrasound, IVU, CT/MRI***.
Ureteric calculus	*Suggested by:* renal colic, moving from loin (upper lateral) down to ***RLQ***, hematuria.
	Confirmed by: ***urinalysis, renal ultrasound, IVU, CT/MRI***.
Appendicitis	*Suggested by:* pain initially central, then radiating to *right* lower quadrant, anorexia, low grade fever, constipation. ***RLQ*** tenderness and guarding.
	Confirmed by: Inflamed appendix at laparotomy
Salpingitis	*Suggested by:* fever, nausea, vomiting, muco-purulent cervical discharge, irregular menses. Bilateral lower abdominal tenderness and guarding.
	Confirmed by: ***CBC:*** leucocytosis. ***High vaginal swab, laparoscopy***.

Acute lower central (hypogastric) abdominal pain

Ulcerative colitis	*Suggested by:* abdominal pain, diarrhea with blood and mucus.
	Confirmed by: ***stool microscopy and culture, colonoscopy***.
Large bowel obstruction	*Suggested by:* severe distension, late vomiting, visible peristalsis, resonant percussion, increased bowel sounds. Supine ***X ray*** showing peripheral abdominal large bowel shadow (with haustra partly crossing the lumen). Fluid levels on erect film.
	Confirmed by: ***abdominal ultrasound and laparotomy findings***.
Cystitis	*Suggested by:* frequency, urgency, dysuria, hematuria.
	Confirmed by: ***urinalysis*** , culture & sensitivity test.
Ectopic pregnancy	*Suggested by:* constant unilateral pain , referred shoulder pain, amenorrhea , vaginal bleeding (usually less than normal period), faintness with an acute rupture.
	Confirmed by: pregnancy test +ve , bimanual examination reveals slightly enlarged uterus, ***pelvic ultrasound*** shows empty uterus.